LETTERS *to the Editor*

Add Fads, Facts, Fundamentals

To the Editor: I agree most heartily with Dr. Mason's [Calif Med 112:61, Jun 1970] suggestion to initiate a column on myths in medicine. I promise that I can keep you supplied with a goodly amount of material. Should you decide to do so how about these for starters?

1. *"Milk makes mucous."* By what physiological mechanism ingestion of milk produces mucous in the respiratory tract is not to be found in any text of physiology nor am I aware of any controlled evidence to suggest such a relationship.

2. *Parentectomy is good treatment for asthmatic children.* That, of all the environmental changes that occur when an asthmatic child is returned from Denver to his smog-laden home, contact with parents should be singled out as the cause for recurrence, seems too far-fetched to be taken seriously.

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Quick Replies

To the Editor: The friendly disagreement between Doctors Perkins, Sahud and myself in regard to certain clotting tests is based mainly on semantics (Calif Med 112:60, June 1970). Thus, I object to their calling certain *limitations* of the one-stage prothrombin time *defects.* The test is so standardized that reproducible results have been obtainable for 42 years but the interpretations of results have greatly altered as newer information has become available. The test is *not* always a measure of prothrombin. For instance, in fresh normal human plasma, the determinant of the prothrombin time is the concentration of the free, not the total, prothrombin[1] and in hereditary hypoprothrombinemia, the test is a reliable quantitative procedure for determining prothrombin. The prothrombin time of a patient on oral anticoagulant therapy, on the contrary, is mainly a measure of factor VII depression. While prothrombin is also reduced, it is so to a lesser extent and in the unmodified test is masked. In hereditary factor VII deficiency, the one-stage test is the only means to detect and measure factor VII since this factor does not participate in intrinsic coagulation. Factor VII is a co-factor of tissue thromboplastin and such terms as convertin or proconvertin are purely speculative. Since bleeding occurs when the concentration of factor VII is reduced below a certain limit, the prothrombin time actually monitors the bleeding propensity. In the control of anticoagulant therapy, the prothrombin time serves as a guide for keeping factor VII above the critical *bleeding* level, but this is not necessarily the *therapeutic level.*

It is certainly not a *defect* of nature that a drug such as Dicumarol® reduces first and foremost factor VII, a factor which plays no role in intrinsic or intravascular clotting but brings about serious bleeding when sufficiently depressed. That the one-stage prothrombin time sensitively measures the depression of factor VII is obviously not a *defect* but a propitious attribute which has saved innumerable patients from bleeding. It is interesting to speculate about what would have happened had Russell viper venom been introduced as the standard throm-

boplastic agent instead of rabbit brain. In the early series of cases with the dosage of Dicumarol controlled by the one-stage test using Russell viper venom, bleeding occurred in 40 percent of the cases.[2] Yet, that the Russell viper venom thromboplastin makes the one-stage prothrombin time sensitive to prothrombin depression can hardly be construed as introducing a *defect*. The misunderstanding lies in our interpretation of terms. *Error, limitation,* and *defect* are not synonymous.

The question of semantics is even more apropos in regard to the term *function* of platelets. Only two *functions* of platelets are experimentally and directly demonstrable: clot retraction and the generation of thromboplastin. When platelets are removed by high centrifugation, the resulting plasma when clotted shows neither retraction nor generation of thromboplastin by the test of Biggs and Douglas or by the prothrombin consumption time. But on adding known quantities of intact platelets to platelet-depleted plasma, both clot retraction and prothrombin consumption become proportional to the number of normal intact platelets.[3] Platelet aggregation, viscous metamorphosis, platelet stickiness, and abnormal morphology are not *functions* but properties of platelets. One may postulate that when platelets have such abnormal properties, they do not function normally but, unfortunately, reliable direct methods to demonstrate the dysfunction are difficult to find. At best, the platelet plug concept of hemostasis is a *theory*.

It should be remembered that serious errors are made by interpreting a laboratory observation in the framework of an expected clinical finding. The reverse is far more trustworthy: correlating the clinical finding with the results of the laboratory tests. I have yet to find a thrombopathy characterized by clinical bleeding in which the prothrombin consumption test carried out by my standardized technique gave normal results.[1]

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3. Quick AJ, Shanberge JN, Stefanini M: The role of platelets in the coagulation of the blood. Amer J Med Sci 217:198, 1949
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The Distribution Of Population Growth

To the Editor: The following table was prepared from the 1970 World Population Sheet compiled by the Population Reference Bureau, Washington, D.C. Fractions have been converted to whole numbers.

Region or Country	*Population Estimates Mid-1970 (millions)*	*Number of Years to Double Population*	*Population Projections to Mid-1985 (millions)*
Latin America	283	24	435
Africa	344	27	530
India	555	27	808
China (Mainland)	760	39	965
U.S.A.	205	70	242
U.S.S.R.	243	70	287
France	51	88	58
Italy	54	88	60
West Germany	59	117	63
U.K.	56	140	62

This data confirms the previously known fact that the rate of population growth is considerably greater in the underdeveloped countries of Latin America, Africa and Asia than in the industrialized countries of Western Europe. While the populations and their rates of growth in the United States of America and the U.S.S.R. are comparable, it is sobering to consider that the population of China will be one thousand five hundred and twenty million by the year 2,009 A.D. The rapid population growth in countries which are relatively poor and least able to absorb the increase in people raises a number of questions. How will the additional people be fed? How can they be educated and what will they do in countries which are currently unable to educate and train their people? Finally, will some of these countries engage in wars of territorial expansion as the classic answer to population increase? A possible solution would be to direct some of the enormous amount of talent, thought, energy, and money at present used to limit the population growth of the United States and Europe towards educating the people of China, India, Africa, Latin America, etc.

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